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ANTHROPOGENIC CHANGE, BIODIVERSITY LOSS, AND A NEW AGENDA FOR EMERGING DISEASES

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ABSTRACT: In this article, we lay out a new agenda for emerging infectious diseases (EIDs). We review their definitions, discuss the emergence of EIDs in wildlife populations, and demonstrate that the common factor defining the group is an overwhelming human basis for their emergence. That is, anthropogenic environmental changes drive EID emergence throughout the human–wildlife–domestic animal continuum and within plant host populations. Classically, parasitic EIDs are among the most significant to affect human, animal, and plant populations. Among these are a series of high-impact wildlife EIDs responsible for mortality of endangered species, host population declines, and species extinctions. These threats to biodiversity are driven by a suite of complex, newly reported, and poorly understood anthropogenic drivers. In this article, we propose a key role for parasitologists in addressing wildlife EIDs in understanding their complexity and, ultimately, in preventing their spread.

DEFINING EMERGING DISEASES

The term "emerging infectious disease" was probably first coined in the second half of the 20th Century. But the term really came to the fore after a series of large outbreaks of infectious diseases in humans during the 1970s and 1980s (Lyme disease, AIDS, antibiotic-resistant microbial infections, legionnaire's disease, and others. Amid calls for increased funding for surveillance programs, the term became widely accepted to represent these new threats to public health (Krause, 1981; Lederberg et al., 1992; Berkelman et al., 1994). The term is now widely used, especially by virologists (e.g., Morse, 1993a; Mahy and Murphy, 1997), and forms the basis for much of the Centers for Disease Control and Prevention's approach to public health in the new millennium (Binder et al., 1999). Definitions of the term "emerging" are given early in the literature (e.g., Krause, 1981; Lederberg et al., 1992). Emerging diseases are those that have recently increased in incidence, impact, or in geographic or host range (e.g., Lyme disease, TB, West Nile virus [WNV], Nipah virus); that are caused by pathogens that have recently evolved (e.g., new strains of influenza virus, drug-resistant strains of malaria); that are newly discovered (e.g., Hendra virus); or are diseases that have recently changed their clinical presentation (e.g., hanta virus pulmonary syndrome). Many authors vary in their definitions of "recent," but most agree that EIDs are those that have emerged within the last 2–3 decades (Lederberg et al., 1992). "Reemerging" diseases are a subclass of emerging diseases that historically occurred at significant levels but which became less significant and only recently have increased in incidence. For the purposes of this article, we follow the definitions listed by Daszak et al. (2000) and consider diseases emerging over the past 3 decades, with reemerging diseases classed simply as EIDs.

WILDLIFE EIDs AND BIODIVERSITY LOSS

Over the past few years, a growing number of infectious disease outbreaks have been reported from wildlife populations. Many of these can be classed as emerging by following the same criteria that define EIDs in human populations (Daszak et al., 2000). These wildlife EIDs have resulted in a series of mass mortalities, population declines, and even extinctions in the last 2 decades. Wildlife EIDs include diseases caused by domestic animal pathogens such as canine distemper virus responsible for mass mortality of African wild dogs and the near-extinction of the North American black-footed ferret. Plague, a disease that still remains enzootic in North America after the last great pandemic in the 1860s, causes mortality as high as 98% in prairie dogs during cyclical outbreaks and was a significant factor in the initial demise of the last wild black-footed ferret population (Thorne and Williams, 1988). A group of newly described morbilliviruses have emerged as significant causes of mortality in marine mammals, along with a range of other marine EIDs, including diseases of hard and soft corals, fibropapillomatosis of sea turtles, rickettsial infections of black abalone, labyrinthulid infection in eel grass, *Perkinsus* infection in oysters, and many others (Harvell et al., 1999; Kim et al., 2003). New infections of garden birds, such as mycoplasmal conjunctivitis in house finches in the United States and salmonellosis in passerines in the U.K., have emerged apparently as a product of the artificially high density and rate of interspecies contact that backyard feeders foster (Fischer et al., 1997; Kirkwood, 1998). Less well-known diseases, such as kangaroo blindness (caused by an orbivirus) or pilchard herpesvirus disease, have emerged over large areas, causing extensive wildlife mortality (Whittington et al., 1997; Hooper et al., 1999). Finally, a group of wildlife EIDs have been cited as the cause of species extinctions. Avian malaria and avian pox are strongly implicated in the extinction of a range of endemic birds of Hawaii (Van Riper et al., 1986), and the first definitively proven case of extinction by infection was caused by a microsporidial infection in a *Partula* sp. snail (Cunningham and Daszak, 1998).

The devastating impact of wildlife EIDs is a product of unusual ecological situations. First, host–parasite ecological theory suggests that it is impossible for a pathogen to cause extinction of its host species or population. This is because the basic reproductive number (R_0–the number of secondary cases caused by a single primary case within a susceptible population) of the parasite is related to host population density. Below a certain population density (the threshold density), R_0 decays to below 1 and the pathogen becomes extinct before the host. However, in many cases, pathogens are cointroduced with hosts in which they have coevolved over many millennia, e.g., introduction of smallpox and measles into the New World. Here, the continued presence of the introduced host (which is often more

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resistant or has a higher herd immunity) heightens the impact on the endemic species.

Second, data suggest that pathogen pollution (see Cunningham et al., 2003) must be a relatively rare event (Mitchell and Power, 2003; Torchin et al., 2003). Invading species often lose pathogens and parasites as they move into new regions, e.g., the European starling introduction into North America (Dobson and May, 1986) and the green crab moving into the New World (Torchin et al., 2002). This situation arises because small numbers of hosts usually move, or are moved, to new regions, reducing the probability of an infected host being in the introduced cohort. Once introduced, invading pathogens must become established to persist (Anderson and May, 1986). If small numbers of hosts are introduced, successful invasion requires an ability to persist at low host density, infect new hosts, or both. However, these problems encountered by introduced pathogens appear to be overwhelmed by the unprecedented magnitude of current trade in wildlife, plants, and their products.

ANTHROPOGENIC ENVIRONMENTAL CHANGE: A COMMON LINK IN THE CHAIN OF EMERGENCE

If we take a broad view of EIDs, we can see that many historical disease epidemics would have been considered emerging. Some authors propose an early phase of disease emergence, whereby pathogens were spread by humans moving across late Pleistocene land bridges (MacPhee and Marx, 1997). The next phase followed the domestication of animals by humans and evolution of measles virus and others (Norrby et al., 1985) that coincided with the building of the first cities and the expansion of human populations above the threshold density for these pathogens (Dobson and Carper, 1996). A third phase occurred during the 14th–17th centuries, with the emergence of pandemic bubonic plague in Europe and the spread of measles, influenza, smallpox, and other agents into the New World (Crosby, 1986). This was driven by advances in maritime technology, globalization of trade, and human population migration across evolutionary boundaries into the New World. Even more recently, the formation of densely packed metropolitan regions during the industrial revolution resulted in another wave of disease emergence. This was followed by the current phase of disease emergence, with antibiotic-resistant microbes, newly emerged zoonoses (e.g., HIV and Nipah virus), a series of pandemic outbreaks driven by unprecedented globalization of human movement and trade, and the centralization of food-processing industries, among others (Morse, 1993b).

Wildlife EIDs are driven by a series of similar processes, including encroachment of humans and domestic animals into wildlife habitat (e.g., canine distemper in African wild dogs), agricultural changes (e.g., transmissible spongiform encephalopathies in zoo animals), anthropogenic introduction of pathogens or "pathogen pollution" (e.g., amphibian chytridiomycosis), and others. Cunningham et al. (2003) comment that some emerging plant diseases are also driven by pathogen pollution. Thus, a common defining theme for all EIDs (of humans, wildlife, domestic animals, and plants) is that they are driven to emerge by anthropogenic changes to the environment. This is essentially a process of natural selection in which anthropogenic environmental changes perturb the host–parasite dynamic equilibrium, driving the expansion of those strains suited to the new environmental conditions and driving the expansion of others into new host species. The selection process acts on the immense pool of varied pathogen strains circulating within the population, c.f., the "zoonotic pool" of Morse (1993b). Thus, very few EIDs are caused by newly evolved pathogens, although notable exceptions include drug-resistant pathogens, newly reassorted influenza strains, and pathogens with point mutations that increase their virulence, e.g., canine parvovirus (Parrish et al., 1985). Even in these examples, it is possible that the new strains were already present in the pathogen population. For example, recent work shows that drug-resistant strains of some common microbes circulate within rodent populations in areas outside the normal contact with antibiotics (Gilliver et al., 1999).

NEW CHALLENGES FOR PARASITOLOGISTS

Drivers of emergence

A series of recent studies have cited new types of anthropogenic environmental changes as the drivers of disease emergence. Ostfeld and Keesing (2000) and Schmid and Ostfeld (2001) demonstrate that reduced reservoir biodiversity correlates with increased risk of Lyme disease transmission to humans and may be a general rule for frequency-dependent transmission. Cunningham et al. (2003) raise awareness of pathogen pollution, the anthropogenic introduction of pathogens across evolutionary and ecological boundaries, as a prime driver of wildlife EIDs. Theirs and other data suggest that Lyme disease emergence was driven by a complex interaction among biodiversity loss, habitat fragmentation, urban sprawl, and the historical shift in agriculture from the eastern United States to the midwest. For other diseases, overexploitation of fisheries (e.g., phocine distemper outbreaks), climate change (e.g., Harvell et al., 2002), deforestation (e.g., malaria and Nipah virus), and pollution (e.g., marine mammal morbilliviruses) have all been cited as drivers of emergence and its importance as a global threat to public health.

Predicting future emergence

The expansion of our earlier anthropocentric focus for EIDs and the identification of new drivers lead us to a number of predictions. First, the rate of emergence of previously unknown zoonotic pathogens will increase. We have shown that anthropogenic disturbance to wildlife habitat may lead to disease emergence. It follows that these changes may also lead to increased transmission of zoonotic pathogens to human populations. Because around 75% of the EIDs that affect humans are zoonotic (Taylor et al., 2001), it is likely that the continual expansion of anthropogenic impact on wildlife habitats will increase the rate of zoonotic emergence.

Second, there will be increasing conflicts between public health and biodiversity conservation. The threat of zoonotic pathogen spread already impedes conservation of the American bison (due to brucellosis), prairie dogs (due to plague), and other wildlife species. As conservation programs expand and the ecological separation among human, domestic animal, and wildlife populations becomes more blurred, these conflicts are likely to increase.

There will be increasing calls for forecasting of emergence.

The most useful response to a newly emerging pathogen is to locally predict it's routes of spread, it's impact, and high-risk populations that will be affected. Current approaches to surveillance and control of a number of EIDs have begun this predictive process. For example, recent forecasts of Potato late blight outbreaks (caused by *Phytophthora infestans*) have been correctly predicted in over 90% of forecasts based on number of prior days of rain (Johnson et al., 1996). Outbreaks of vector-borne diseases such as Rift Valley fever have also been successfully predicted using data on the El Niño Southern Oscillation (Linthicum et al., 1999). A program has recently been set up to predict emergence of coral bleaching events based on sea temperature increases (Strong et al., 1998). However, the complexities that underlie emergence and a poor understanding of many drivers hinder this work.

There will be a rise in the number of local and species extinctions for which disease emergence is cited as the cause. This rise is likely to be partly due to increased interest in investigating infectious diseases as causes of mortality events and partly due to increasing anthropogenic pressure on wildlife populations.

The expanding threat of pathogen pollution

Pathogen pollution will increasingly drive disease emergence in human, wildlife, and other populations. The recent emergence of WNV in the United States and foot-and-mouth disease (FMD) in the U.K. highlights the continuing threat of disease introduction to public and domestic animal health. Cunningham et al. (2003) comment that most cases of pathogen pollution are related to trade or travel. Examples include pandemic influenza virus infection in humans, HIV emergence in the United States, WNV, FMD, whirling disease of salmonid fish, dutch elm disease, and chestnut blight (Auerbach et al., 1984; Lederberg et al., 1992; Morse, 1993a; Andree et al., 1999; Lanciotti et al., 1999; Samuel and Knowles, 2001; Cunningham et al., 2003). Trade and travel are in turn related to increasing rates of international air transport. Examination of air travel industry data shows that global air travel volume doubled between 1985 and 1996 (Fig. 1) and that the number of new routes offered has increased in direct proportion to this growth (The Boeing Company, 2000). Air travel has accounted for an increasing share of the global gross domestic product (GDP) during the past 16 yr, and projections suggest that travel share of GDP will rise steadily for the next 20 yr (The Boeing Company, 2000). The global air fleet is expected to double in the next 20 yr, whereas world air traffic will grow by 4.9% annually over the next 20 yr, 2 percentage points faster than the predicted global mean annual growth in GDP over the same period (The Boeing Company, 2002). Because this accelerating expansion of air travel is not mirrored by an expansion of disease surveillance budgets (particularly for wildlife EIDs), the impact of these trends on pathogen spread is likely to be significant.

Countering the EID threat

How do we counter emerging diseases? We have shown that disease emergence is an ecological process underpinned by environmental changes that can be identified and measured. We propose a new agenda for EIDs that tackles emergence at its root by examining how anthropogenic environmental changes alter disease ecology. Previous work on disease emergence has largely consisted of outbreak investigation and identification of at-risk populations, key drivers, and modes of spread, followed by measures to combat the current outbreak. We propose that, by a series of research efforts focused on the root causes of EIDs, it will be increasingly feasible to understand common themes of emergence, to identify high-risk human activities that foster emergence, and to predict future emergence of novel agents (as opposed to forecasting outbreaks of known pathogens—see above).

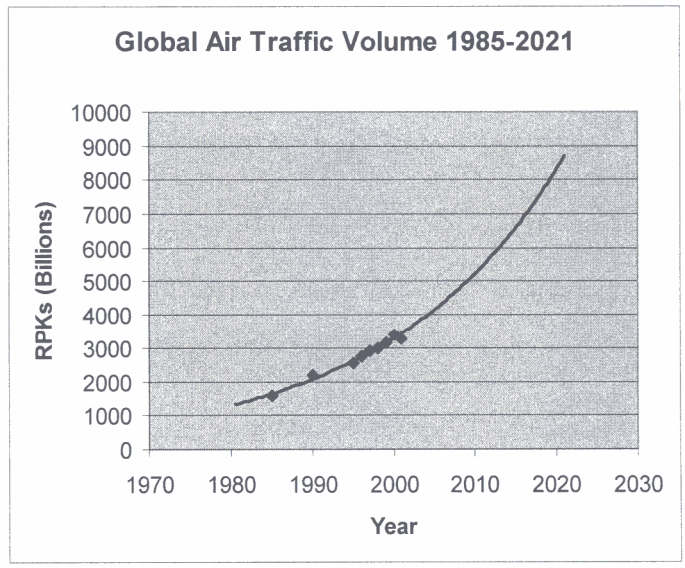

FIGURE 1. Volume of global air traffic, 1985–2001, and projection of future trends from 2001 to 2021. These data demonstrate that global air traffic volume is undergoing an accelerating expansion that will continue for the next 2 decades. The chart is based on air industry data (The Boeing Company, 2002) and plots the volume of human air traffic measured in revenue passenger kilometers (RPKs)—the total number of passengers traveling globally multiplied by the number of kilometers they fly.

Some authors (e.g., Murphy, 1998) previously have suggested that prediction on this scale will be very difficult, considering that the biodiversity of agents remaining to be discovered. However, for some pathogens with restricted reservoir hosts, it is possible to predict their biodiversity. For example, Telford et al. (1997) used information on guilds of deer tick–transmitted zoonotic pathogens in Eurasian *Ixodes* spp. ticks with those described from America to target surveillance, resulting in the discovery of a novel flavivirus, "deer tick virus," related to the virulent Powassan virus. Nipah, Hendra, and Menangle viruses are paramyxoviruses that have recently been identified within fruit bat reservoir hosts in Australia and Malaysia and emerge through domestic animal amplifier hosts (Field et al., 2001). The biodiversity of fruit bats is largely known and clearly defined, with many endemic species on oceanic islands. A logical approach would be to investigate the biodiversity of these viruses in fruit bats throughout their range and then introduce control measures where domestic animals co-occur.

There is a clear role for parasitologists in this new, bottom-up approach to EIDs. Parasitic diseases are classical EIDs in human populations, e.g., toxoplasmosis, cryptosporidiosis, drug-resistant malaria, malarial reemergence in montane re-

gions. Eukaryotic parasites are also among the most significant causative agents of wildlife EIDs. For example, most of the diseases implicated in wildlife species extinctions are caused by pathogens classically studied by parasitologists, e.g., avian malaria, amphibian chytridiomycosis, microsporidiosis in *Partula* sp. snails, and the slime mold *Labyrinthula* (Daszak and Cunningham, 1999). Parasitologists have probably made the most significant advances in understanding disease ecology in wild animals. Finally, parasitologists are well-versed in the biocomplexity of disease biology, through elucidation of multiple host life cycles.

Even now, more than 10 yr after the publication of the Institute of Medicines report on emerging infections (Lederberg et al., 1992), diseases continue to emerge in human and wildlife populations. A new approach is required, a new collaboration between parasitologists and the other disciplines involved in understanding anthropogenic change and disease emergence.

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